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Title:

Post-exercise glucose-fructose co-ingestion augments cycling capacity during short-term and overnight recovery from exhaustive exercise, compared to isocaloric glucose.

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Fructose co-ingestion augments overnight recovery

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36

Abstract

During short-term recovery, post-exercise glucose-fructose co-ingestion can accelerate total glycogen repletion and augment recovery of running capacity. It is unknown if this advantage translates to cycling, or to a longer (e.g. overnight) recovery. Using two experiments, the present research investigated if post-exercise glucose-fructose co-ingestion augments exercise capacity following 4 h (SHORT-EXPERIMENT; $n=8$) and 15 h (OVERNIGHT-EXPERIMENT; $n=8$) recoveries from exhaustive exercise in trained cyclists, compared to isocaloric glucose alone. In each experiment, a glycogen depleting exercise protocol was followed by a 4 h recovery, with ingestion of 1.5 or 1.2 g.kg⁻¹.h⁻¹ carbohydrate in SHORT-EXPERIMENT (double-blind) and OVERNIGHT-EXPERIMENT (single-blind), respectively. Treatments were provided in a randomized order using a cross-over design. Four or fifteen hours after the glycogen depletion protocol, participants cycled to exhaustion at 70 or 65% W_{\max} in SHORT-EXPERIMENT and OVERNIGHT-EXPERIMENT, respectively. In both experiments there was no difference in substrate oxidation or blood glucose and lactate concentrations between treatments during the exercise capacity test (trial effect, $p > 0.05$). Nevertheless, cycling capacity was greater in GLUCOSE+FRUCTOSE *versus* GLUCOSE-ONLY in SHORT-EXPERIMENT (28.0 ± 8.4 *versus* 22.8 ± 7.3 min, $d = 0.65$, $p = 0.039$) and OVERNIGHT-EXPERIMENT (35.9 ± 10.7 *versus* 30.6 ± 9.2 min, $d = 0.53$, $p = 0.026$). This is the first study to demonstrate that post-exercise glucose-fructose co-ingestion enhances cycling capacity following short-term (4 h) and overnight (15 h) recovery durations. Therefore, if multi-stage endurance athletes are ingesting glucose for rapid post-exercise recovery then fructose containing carbohydrates should be co-ingested.

62 **Keywords:** Exercise; Fructose; Glucose; Metabolism; Nutrition

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Introduction

Carbohydrate is the dominant energy source during moderate-to-high-intensity exercise (Romijn et al., 1993). Human carbohydrate stores are finite, with skeletal muscle and liver storing ~500 g (Jensen et al., 2011) and ~100 g of glycogen (Taylor et al., 1996), respectively. During moderate-to-high-intensity exercise, total carbohydrate oxidation exceeds maximum exogenous carbohydrate oxidation. Therefore, during prolonged exercise, low glycogen availability is inevitable (King et al., 2018; Tsintzas et al., 2001). Low glycogen availability is associated with the onset of fatigue, demonstrated by the strong relationship between muscle (Bergstrom et al., 1967) and liver (Casey et al., 2000) glycogen content and exercise capacity. Consequently, for optimal performance, endurance athletes should aim to maximize pre-exercise glycogen content. Moreover, in repeated endurance bouts with short recovery duration the rate of glycogen repletion is the principal determinant of recovery time (Alghannam et al., 2016; Casey et al., 2000).

To maximize glycogen repletion, guidelines recommend ingesting 1.0-1.2 g.kg⁻¹.h⁻¹ carbohydrate of moderate-to-high glycemic index during the first 4 h of recovery, before resuming daily fuel needs (Burke et al., 2011). These guidelines are primarily based on evidence relating to maximizing muscle glycogen repletion (Betts and Williams, 2010). However, liver glycogen is also important as it contributes ~17% to total glycogen stores and contributes to the maintenance of blood glucose availability during exercise, a lack of which is associated with the onset of fatigue (Coyle et al., 1984). In contrast to muscle glycogen, liver glycogen synthesis is potentially increased by co-ingestion of the low-glycemic index carbohydrate, fructose, with glucose (or

sucrose ingestion; a glucose-fructose disaccharide), when compared to isocaloric glucose alone (Decombaz et al., 2011; Fuchs et al., 2016; 2019). Moreover, glucose-fructose co-ingestion does not compromise muscle glycogen repletion (Wallis et al., 2008), resulting in greater total glycogen storage, compared to isocaloric glucose (Fuchs et al., 2016). Maunder et al. (2018) is the only study to investigate if this translates into improved exercise capacity when ingesting large quantities of carbohydrates ($>1.2 \text{ g.kg}^{-1}.\text{h}^{-1}$), reporting 90 g.h^{-1} fructose-maltodextrin (glucose polymer) co-ingestion augments 4-h recovery of running capacity compared to maltodextrin alone.

Muscle glycogen depletion is associated with fatigue in running, whereas hypoglycemia (indicative of liver glycogen depletion) is more implicated in fatigue during cycling (Tsintzas and Williams, 1998). Therefore, it cannot be assumed that accelerated recovery of running capacity with post-exercise maltodextrin-fructose co-ingestion (Maunder et al., 2018) translates to cycling. Furthermore, the relatively short recovery period in the prior study (4-h) may have resulted in the ingested carbohydrate being unabsorbed at the start of exercise. Indeed, the greater exercise capacity with maltodextrin-fructose co-ingestion may be partially attributed to faster intestinal absorption of glucose-fructose *versus* glucose alone (Jentjens and Jeukendrup, 2005) enabling greater short-term carbohydrate availability. Consequently, it is unknown if enhanced exercise capacity is generalizable to an overnight recovery, relevant to multi-day events (e.g. Grand Tours). Therefore, the aim of the present experiments was to determine whether post-exercise glucose-fructose co-ingestion improves exercise capacity following 4 h (SHORT-EXPERIMENT) and 15 h (OVERNIGHT-EXPERIMENT) recoveries from exhaustive exercise in trained cyclists. We

113 hypothesized that, compared to glucose alone, isocaloric glucose-fructose co-
114 ingestion would augment cycling capacity in SHORT- and OVERNIGHT-
115 EXPERIMENT.

Methods

Study design

The present research comprised two randomized, cross-over experiments conducted at the University of Bath, investigating the effect of post-exercise glucose-fructose co-ingestion *versus* glucose alone on subsequent cycling capacity. Experiment one (double-blind) employed a 4-h recovery (SHORT-EXPERIMENT), whereas, experiment two (single-blind) used a 15-h overnight recovery (OVERNIGHT-EXPERIMENT). Experimental procedures were approved by the University of Bath Research Ethics Approval Committee for Health (SHORT-EXPERIMENT: MSES 17/18-007, OVERNIGHT-EXPERIMENT: MSES 17/18-006), conducted according to the Declaration of Helsinki, and participants provided informed, written consent prior to participation.

Participants

Eight trained male cyclists participated in SHORT-EXPERIMENT (**Table 1**). Three female and five male trained cyclists participated in OVERNIGHT-EXPERIMENT. Volunteers were recruited from local cycling and triathlon clubs. Inclusion criteria included: weekly aerobic training >3 h and $\dot{V}O_{2peak} >45 \text{ ml.kg}^{-1}.\text{min}^{-1}$.

139

140 *Preliminary Testing*

141 $\dot{V}O_{2peak}$ and peak power (W_{max}) were determined on an electronically-braked cycle
142 ergometer (Lode Excalibur Sport, Groningen, Netherlands). Power output began at
143 100 W (50 W for females), increasing by 1 W every 3 s until exhaustion. Expired gas
144 was measured continuously using a breath-by-breath analyzer (ParvoMedics
145 TrueOne® 2400, Utah, USA). $\dot{V}O_{2peak}$ was defined as the highest 30 s $\dot{V}O_2$ rolling
146 average.

147

148 *Experimental Trials*

149 SHORT-EXPERIMENT used a full protocol familiarization between preliminary testing
150 and experimental trials. OVERNIGHT-EXPERIMENT used an exercise capacity test
151 familiarization following the $\dot{V}O_{2peak}$ test after a 30-min rest.

152

153 All visits were separated by ≥ 4 days. For females, visits were standardized to the same
154 menstrual cycle phase (by self-report), controlling for the influence of hormonal
155 variance on performance (Julian et al., 2017). Participants refrained from high-intensity
156 exercise for 48 h, and moderate-intensity exercise, caffeine and alcohol for 24 h,
157 before each trial.

158

159 SHORT-EXPERIMENT began with a glycogen depletion protocol (GDP). Participants
160 alternated between 2 min efforts at 90% W_{max} and 50% W_{max} . When 90% efforts could

no longer be sustained, the high intensity effort was reduced to 80% and the alternating process continued. When 80% efforts could not be maintained the alternating process was repeated until exhaustion was reached at 70% W_{max} . OVERNIGHT-EXPERIMENT began between 14:30 and 16:30 with a similar GDP, however, when 70% W_{max} could not be maintained participants continued cycling to complete exhaustion at 50% W_{max} . Both protocols are efficacious at inducing liver and muscle glycogen depletion (Decombaz et al., 2011; Fuchs et al., 2016). Participants were fan cooled and provided water *ad libitum*.

The GDP was followed by 4 h ingesting 1.5 g.kg⁻¹.h⁻¹ or 1.2 g.kg⁻¹.h⁻¹ carbohydrate in SHORT-EXPERIMENT and OVERNIGHT-EXPERIMENT, respectively. In SHORT-EXPERIMENT, after 4 h of recovery, participants cycled to exhaustion at 70% W_{max} . In OVERNIGHT-EXPERIMENT, participants cycled to exhaustion at 65% W_{max} the following morning, 15 h after GDP cessation. Exercise capacity time was recorded upon first attainment (i.e. no three-strike rule) of volitional exhaustion. Verbal encouragement was not provided.

During the exercise capacity test 1-min expired gas samples were collected every 15 min, and at exhaustion. At these timepoints, capillary fingertip measurements of blood glucose (Freestyle Optium, Abbott Diabetes Care, Witney, UK) and lactate (Lactate Pro, Arkray, Kyoto, Japan) concentrations were taken using portable analyzers (typical error ± 0.2 mmol.l⁻¹). In SHORT-EXPERIMENT this was complemented with a 10-cm, 16-item visual analogue scale (VAS) of upper, lower and central gastrointestinal

symptoms. In SHORT-EXPERIMENT, all measures were also taken hourly during the 4-h recovery.

Carbohydrate Test Drinks and Dietary Intake

In SHORT-EXPERIMENT, a self-selected breakfast was replicated prior to each trial. In OVERNIGHT-EXPERIMENT, participants replicated a low-carbohydrate diet ($1.3 \pm 0.8 \text{ g.kg}^{-1}$) prior to the GDP.

In SHORT-EXPERIMENT participants ingested $1.5 \text{ g.kg}^{-1}.\text{h}^{-1}$ of either maltodextrin or sucrose (glucose-fructose disaccharide) in 20% solutions every 30 min during the 4 h recovery. OVERNIGHT-EXPERIMENT ingested $1.2 \text{ g.kg}^{-1}.\text{h}^{-1}$ of either maltodextrin or maltodextrin-fructose (1:1) in hourly 20% solutions for 4 h post-GDP. Hydrolysis of maltodextrin and sucrose is rapid, hence, these carbohydrates are not digested or absorbed at different rates to their constituent monosaccharides (Gray et al., 1966; Hawley et al., 1991). Therefore, hereafter treatments are referred to as GLUCOSE-ONLY and GLUCOSE+FRUCTOSE.

In OVERNIGHT-EXPERIMENT participants consumed a standardized meal during the first hour after the GDP (Energy: 892 kJ, Fat: 11.7 g, Carbohydrate: 19.4 g, Protein: 5.8 g) and the following morning at least 1 h before exercise (Energy: 1784 kJ, Fat: 32.8 g, Carbohydrate: 7.8 g, Protein: 25.0 g). Thus, participants ingested $359 \pm 44 \text{ g}$ carbohydrate between the GDP and the exercise capacity test. Ingesting a low-

carbohydrate meal before exercise removed the influence of exogenous carbohydrate on exercise capacity.

Substrate Oxidation

Expired gas was collected using the Douglas bag technique (Hans Rudolph, Missouri, USA; Servomex MiniMP 5200, Crowborough, UK). During sampling, concurrent measures of inspired air were made to correct for changes in ambient O₂ and CO₂ (Betts and Thompson, 2012). Before each trial the Servomex was calibrated to two gases of known concentrations, zero gas (100% nitrogen) and a mixed gas (16.12% O₂ and 5.06% CO₂). Rates of substrate oxidation were calculated using the moderate- to high-intensity indirect calorimetry equations of Jeukendrup and Wallis (2005).

Statistical Analysis

A priori sample size was calculated using the running capacity data ($d = 1.84$) from Maunder et al. (2018). N=5 would provide 95% statistical power with $\alpha=0.05$. Therefore, eight participants were recruited in each experiment to ensure ample data sets.

In SHORT- and OVERNIGHT-EXPERIMENT exit interviews indicated one and three participants, respectively, were not successfully blinded to treatment. In each experiment, one participant was metabolically unstable during the exercise capacity test (blood lactate concentration $>10 \text{ mmol.L}^{-1}$) and were excluded from metabolite

and substrate oxidation data, as indirect calorimetry is not accurate above lactate threshold (Jeukendrup and Wallis, 2005).

Experimental data were processed and analyzed using Microsoft Excel (Microsoft, Washington, USA) and SPSS v22 (IBM, New York, USA). Differences in gastrointestinal discomfort, blood glucose, lactate and rates of carbohydrate and fat oxidation were assessed between treatments and across time using a two-way repeated measures ANOVA. Following a significant interaction, Bonferroni post-hoc tests were used to locate differences. For all comparisons $\alpha \leq 0.05$. The magnitude of differences between variables were assessed using Cohens *d* effect sizes (*d*). Data are presented as mean \pm SD alongside 95% confidence intervals (95%CI).

Results

SHORT-EXPERIMENT

No differences were detected in GDP duration between GLUCOSE-ONLY and GLUCOSE+FRUCTOSE (61.8 ± 13.5 versus 64.1 ± 10.0 min; $p = 0.44$). No trial order effects were observed for exercise capacity ($p = 0.83$). Exercise capacity was greater in GLUCOSE+FRUCTOSE compared to GLUCOSE-ONLY (28.0 ± 8.4 versus 22.8 ± 7.3 min; $p = 0.039$; **Figure 1A**). This difference was robust to the inclusion ($\Delta 5.1$ min; $d = 0.65$) or exclusion ($\Delta 4.4$ min; $d = 0.54$) of the participant who correctly identified the trial order.

Blood Metabolites

During recovery, blood glucose concentrations increased to a peak at ~60 min before decreasing to baseline concentrations (time effect, $p < 0.01$), with no differences between trials during recovery or during the exercise capacity test (trial effect, $p = 0.37$; time x trial interaction effect, $p = 0.54$; **Figure 2A**).

Blood lactate concentrations were higher in GLUCOSE+FRUCTOSE, compared to GLUCOSE-ONLY (trial effect, $p = 0.01$), which was most apparent in the early phase of recovery (time x trial interaction effect, $p < 0.01$; **Figure 2B**). No differences in blood lactate concentrations were detected during the exercise capacity test (**Figure 2B**).

Substrate Metabolism

Rates of carbohydrate oxidation during the entire recovery period were higher in GLUCOSE+FRUCTOSE *versus* GLUCOSE-ONLY (trial effect, $p = 0.02$; time x trial interaction effect, $p = 0.99$; **Figure 2C**). However, no differences in carbohydrate oxidation between trials were apparent during the exercise capacity test (**Figure 2C**), nor were any differences detected in rates of lipid oxidation during recovery or during the exercise capacity test (trial effect, $p = 0.20$; **Figure 2D**). Whilst a time x trial interaction effect was detected for lipid oxidation rates, no *post-hoc* comparison remained significant following correction for multiple comparisons (all $p > 0.05$).

Gastrointestinal symptoms

Ratings of upper (**Figure 3A**), lower (**Figure 3B**) and central/other gastrointestinal symptoms (**Figure 3C**) all remained low and did not differ between trials (time effect, $p > 0.05$ for all; trial effect, $p > 0.05$ for all; trial x time interaction effect, $p > 0.05$ for all).

OVERNIGHT-EXPERIMENT

There was no difference in GDP duration between GLUCOSE-ONLY and GLUCOSE+FRUCTOSE (96.0 ± 24.2 *versus* 92.2 ± 26.8 min; $p = 0.44$). No trial order effects were detected for exercise capacity ($p = 0.48$). Exercise capacity was greater in GLUCOSE+FRUCTOSE compared to GLUCOSE-ONLY (35.9 ± 10.7 *versus* 30.6 ± 9.2 min; $p = 0.026$; **Figure 1B**). This difference was similar in participants successfully blinded to treatment ($n=5$; $\Delta 4.4$ min; $d = 0.51$) and those who were not ($n=3$; $\Delta 6.7$ min; $d = 0.63$).

285

286 ***Blood Metabolites***

287 Blood glucose concentrations rose across the exercise capacity test (time effect, $p =$
288 0.004), with no differences between trials (trial effect, $p = 0.11$; time x trial interaction
289 effect, $p = 0.74$; **Figure 4A**). This response was mirrored in blood lactate
290 concentrations (time effect $p = 0.004$), with no differences between trials (trial effect,
291 $p = 0.12$; time x trial interaction effect, $p = 0.84$; **Figure 4B**).

292

293 ***Substrate Oxidation***

294 Rates of carbohydrate (**Figure 4C**) and fat oxidation (**Figure 4D**) were stable across
295 time (time effect, $p > 0.05$), with no differences between trials (trial effect, $p > 0.05$;
296 time x trial interaction effect, $p > 0.05$).

Discussion

The present experiments demonstrate that post-exercise glucose-fructose co-ingestion, compared to isocaloric glucose alone, augments cycling capacity following short (4-h) and overnight (15-h) recovery periods in trained cyclists. These data provide functional relevance to previous work, showing that enhancement of post-exercise liver glycogen repletion and short-term recovery of running capacity, translate into improved cycling capacity over 4-h and overnight recovery durations.

Previous research exploring post-exercise glucose-fructose co-ingestion versus glucose alone has investigated relatively short recovery periods <5 h (Casey et al., 2000; Decombaz et al., 2011; Fuchs et al., 2016; Maunder et al., 2018). Relevant to multi-day endurance athletes, we demonstrate that post-exercise glucose-fructose co-ingestion augments cycling capacity after an overnight (15-h) recovery. Exercise capacity tests have been criticized for poor reliability and ecological validity (Currell et al., 2008). However, in races the intensity is often set by the fastest athlete with others maintaining this pace until it becomes unsustainable, hence, exercise capacity tests are relevant to real-world endurance racing (Alghannam et al., 2014).

The improvement in exercise capacity in SHORT-EXPERIMENT ($27.2 \pm 27.5\%$; $d = 0.65$) and OVERNIGHT-EXPERIMENT ($18.0 \pm 19.4\%$; $d = 0.53$) was smaller than that observed by Maunder et al. (2018; $32.4 \pm 19.9\%$; $d = 1.84$). The difference in the magnitude of improvement could be explained by differences in exercise modality or intensity. Debilitating gastrointestinal symptoms are more common in runners than cyclists (Peters et al., 2000). Indeed, in SHORT-EXPERIMENT we observed no

differences in gastrointestinal symptoms between treatments, whereas Maunder et al. (2018) report trends towards greater gastrointestinal discomfort in the glucose treatment. Even mild gastrointestinal distress is associated with impaired endurance performance (O'Brien et al., 2011; Rowlands et al., 2012). Thus, differences in gastrointestinal comfort could explain the smaller difference in exercise capacity compared to Maunder et al. (2018). Furthermore, the present experiments utilized a higher exercise intensity than Maunder et al. (2018; ~80 *versus* 70% $\dot{V}O_{2peak}$), which may have altered the cause of fatigue. Indeed, we found carbohydrate oxidation rates were maintained at exhaustion, whereas, Maunder et al. (2018) report declining rates of carbohydrate oxidation. Thus, in Maunders' research carbohydrate availability (as a fuel) being a more direct cause of fatigue could also partly explain the greater difference in exercise capacity.

The greater improvement in exercise capacity with glucose-fructose co-ingestion in Maunder et al. (2018) compared to OVERNIGHT-EXPERIMENT could further be explained by differences in recovery duration (4-h *versus* 15 h, respectively). It has been suggested that a higher liver glycogen concentration stimulates glycogenolysis (Roden et al., 2001). Therefore, a potential increase in liver glycogen content with glucose-fructose co-ingestion may have diminished overnight. Further contributing to the smaller difference in exercise capacity in OVERNIGHT-EXPERIMENT could be removal of the temporary advantage provided by residual carbohydrate in the gut at exercise initiation. Indeed, intestinal absorption of glucose occurs at ~1 g.min⁻¹ (Jeukendrup et al., 2004), hence the carbohydrate ingested in OVERNIGHT-EXPERIMENT (359 ± 44 g) would have been absorbed in <6 h, glucose-fructose being faster still (Jentjens et al., 2004). Thus, unlike research employing a short recovery,

exercise capacity in OVERNIGHT-EXPERIMENT is more likely to only be influenced by endogenous carbohydrate availability, reducing any metabolic advantage provided through enhanced exogenous carbohydrate availability following glucose-fructose co-ingestion.

In cycling, hypoglycemia is frequently observed at exhaustion (Casey et al., 2000; Coyle et al., 1986), commonly attributed to a reduction in hepatic glucose output due to liver glycogen depletion (Gonzalez et al., 2018). In contrast, in all treatments in the present experiments, blood glucose concentrations *rose* at exhaustion. This could be explained by the high exercise intensity ($\sim 80\% \dot{V}O_{2peak}$). During high intensity exercise epinephrine is the most potent regulator of hepatic glucose output (Marliss et al., 1992; Sigal et al., 1996). Epinephrine increases hepatic glucose output while reducing muscle glucose uptake (Howlett et al., 1999). Consequently, increased epinephrine concentrations at exhaustion (Kreisman et al., 2000) could explain the increase in blood glucose concentration. In the present experiments, high rates of carbohydrate oxidation were maintained at exhaustion, which contrasts with prior work in running (Mauder et al., 2018). This, in addition to the lower total carbohydrate utilized during the exercise capacity tests in the present experiments *versus* that of Mauder et al. (2018; <130 *versus* >157 g), suggests that low carbohydrate availability may have been less important as a fatigue mechanism in the present experiments. Nevertheless, it is plausible that even a relatively small decrease in carbohydrate oxidation rates could have contributed to fatigue, due to the high exercise intensity. Furthermore, it is possible that enhanced liver glycogen content after glucose-fructose co-ingestion facilitated the greater exercise capacity *via* mechanisms other than providing fuel. Indeed, greater carbohydrate availability is associated with a reduction in perceived

effort for a given exercise intensity (Marquet et al., 2016; Noakes et al., 2004). However, it was not possible to measure liver glycogen content in the present experiments, and therefore it cannot be determined directly if there was greater liver glycogen availability after glucose-fructose co-ingestion. Nevertheless, we did observe increases in carbohydrate oxidation and blood lactate concentrations at rest following glucose-fructose co-ingestion *versus* glucose alone, which is consistent with the hypothesis of greater liver glycogen repletion and hepatic conversion of fructose to lactate (Fuchs et al., 2016).

In conclusion, post-exercise glucose-fructose co-ingestion augments cycling capacity following short (4-h) and overnight (15-h) recoveries from exhaustive exercise, compared to isocaloric glucose alone. Therefore, endurance athletes competing in multi-stage events where recovery time is limited may benefit from consuming fructose alongside glucose in their post-exercise nutritional strategies.

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Table 1. Participant Characteristics.

	SHORT-EXPERIMENT	OVERNIGHT-EXPERIMENT
Age (years)	24 ± 7	24 ± 5
Mass (kg)	73.2 ± 7.6	69.1 ± 9.1
Height (m)	1.81 ± 0.08	1.78 ± 0.09
$\dot{V}O_{2peak}$ (mL.kg ⁻¹ .min ⁻¹)	66.9 ± 6.1	62.9 ± 9.8
W_{max} (W)	403 ± 34	378 ± 74

525 Data are presented as mean ± SD.

526

527

Figure Legends

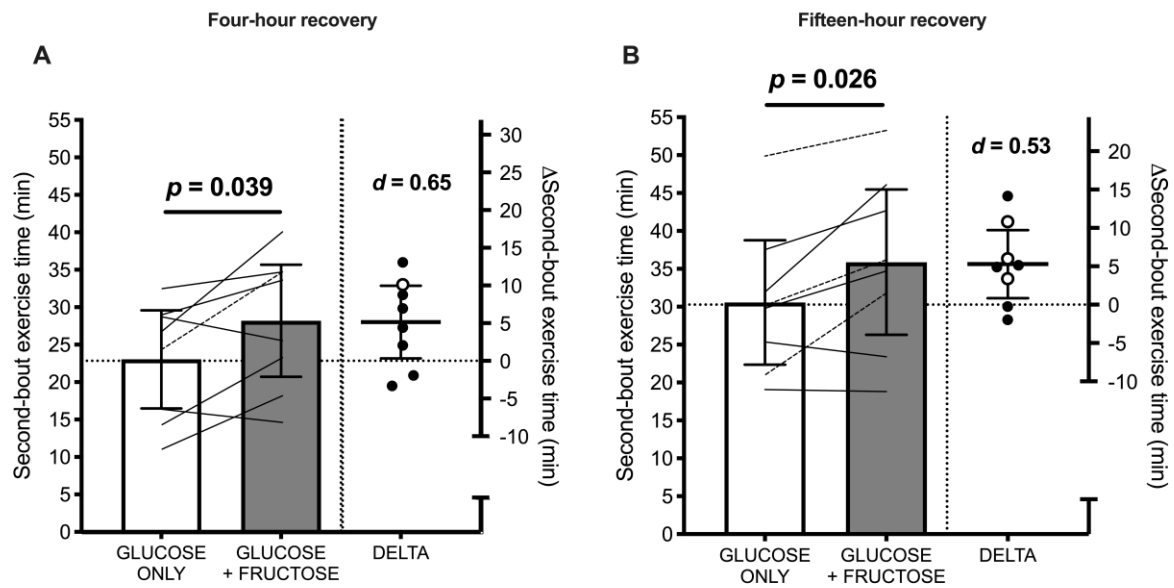


Figure 1. Exercise capacity following 4-h (SHORT-EXPERIMENT: **A**) and 15-h (OVERNIGHT-EXPERIMENT: **B**) recovery from exhaustive exercise, ingesting glucose alone (GLUCOSE-ONLY) or isocaloric glucose plus fructose co-ingestion (GLUCOSE+FRUCTOSE), in trained cyclists. Bars and horizontal lines represent means \pm 95%CI ($n=8$ in both experiments). Lines and circles show individual responses, with those who were unsuccessfully blinded to treatment displayed as dashed lines or open circles, respectively.

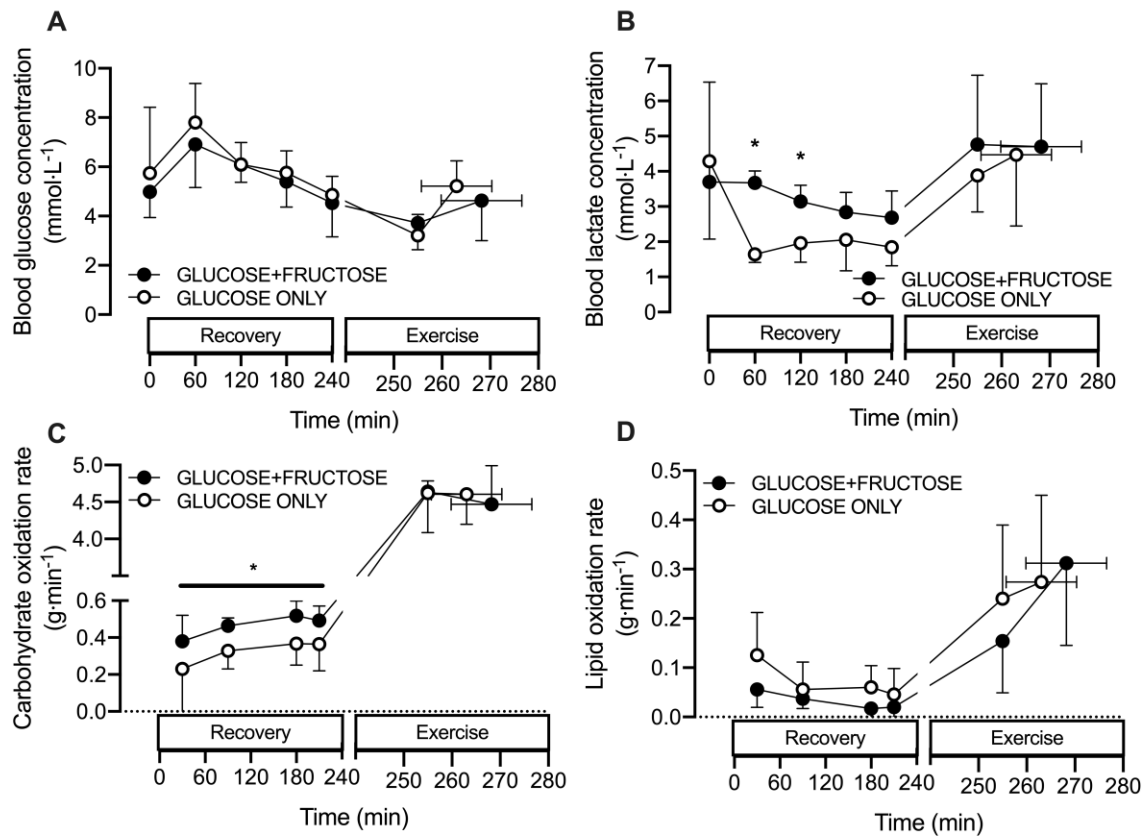


Figure 2. Blood glucose concentrations (**A**), blood lactate concentrations (**B**), carbohydrate oxidation (**C**) and fat oxidation (**D**) responses during 4-hours of recovery and a subsequent exercise capacity test with ingestion of glucose alone (GLUCOSE-ONLY) or isocaloric glucose plus fructose co-ingestion (GLUCOSE+FRUCTOSE) in trained cyclists ($n=7$). Data are presented as mean \pm 95% CI. *denotes a significant trial effect ($p < 0.05$).

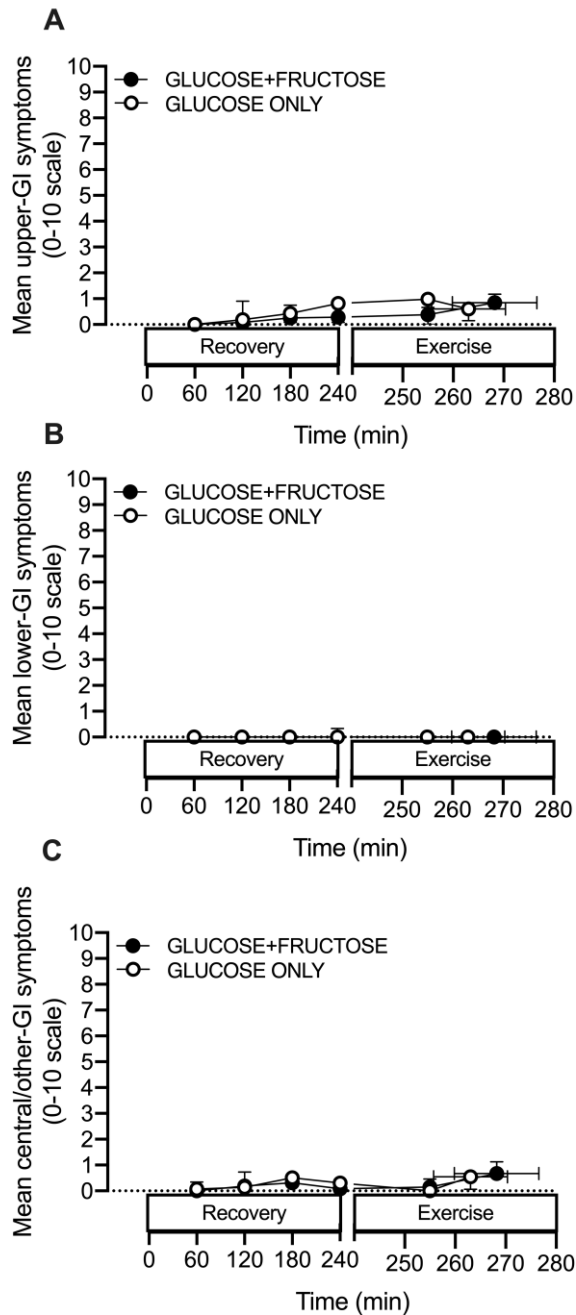


Figure 3. Upper (A), lower (B) and central/other (C) gastrointestinal symptoms during 4-hours of recovery and a subsequent exercise capacity test with ingestion of glucose alone (GLUCOSE-ONLY) or isocaloric glucose plus fructose co-ingestion (GLUCOSE+FRUCTOSE) in trained cyclists ($n=7$). Data are presented as mean \pm 95% CI. *denotes a significant trial effect ($p < 0.05$).

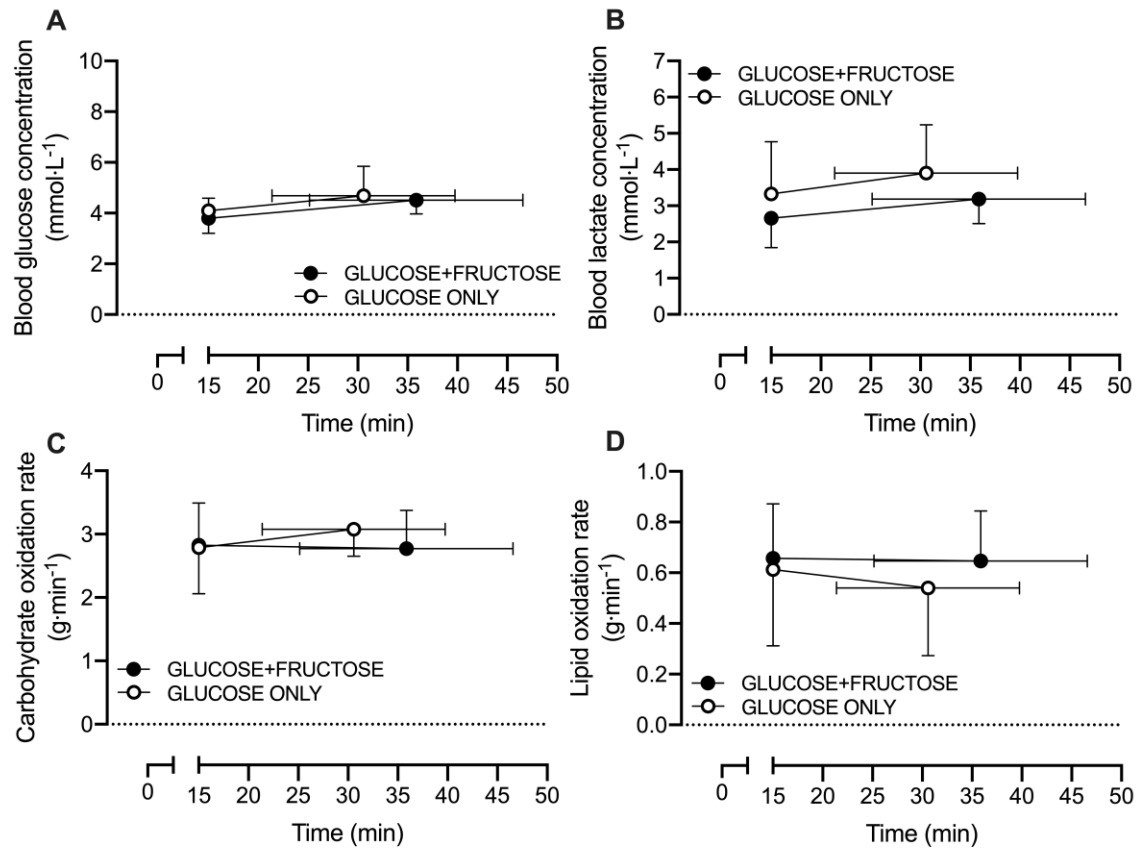


Figure 4. Blood glucose concentrations (**A**), blood lactate concentrations (**B**), carbohydrate oxidation (**C**) and fat oxidation (**D**) responses after 15-h recovery during the exercise capacity test with ingestion of glucose alone (GLUCOSE-ONLY) or isocaloric glucose plus fructose co-ingestion (GLUCOSE+FRUCTOSE) in trained cyclists ($n=7$). Data are presented as mean \pm 95%CI. * denotes a significant trial effect ($p < 0.05$).

